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5: [Ahmed M, Cramer SD, Lyles DS.](#)

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J Neurosurg. 2004 Jun;100(6):1049-59.  
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█ Vesicular stomatitis virus expressing a chimeric Sindbis glycoprotein containing an Fc antibody binding domain targets to Her2/neu overexpressing breast cancer cells.

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█ VSV strains with defects in their ability to shutdown innate immunity are promising systemic anti-cancer agents.

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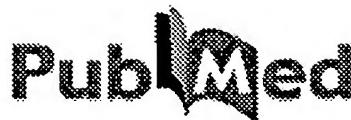
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- 20:** [Balachandran S, Porosnicu M, Barber GN.](#) [Related Articles.](#)
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**Exploiting tumor-specific defects in the interferon pathway with a previous unknown oncolytic virus.**

*Nat Med.* 2000 Jul;6(7):821-5.

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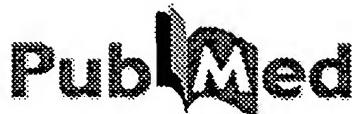
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## Oncogenes-antioncogenes and virus therapy of cancer.

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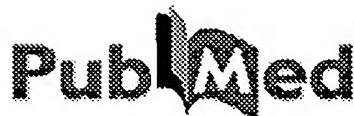
Department of Medicine, University of South Florida College of Medicine, Tampa.

Viruses can render services to mankind. 1. Retroviruses pinpoint and transduce cellular oncogenes. 2. Retroviral vectors can introduce antioncogenes (the R1 gene) into malignant cells thus rendering the recipient cells nonmalignant. 3. Oncolytic viruses lyse tumor cells. 4. Parvoviruses replicate only in dividing and exert lysis and antioncogene effect in tumor cells without affecting restir normal cells. 5. Myxo- and paramyxoviruses (and other viruses) upgrade the immunogenicity of cell surface antigens thus eliciting rejection type host immunity against these cells which is operational against not virus-infected cells of the same type (post-oncolytic antitumor immunity). 6. Viruses or virally infected cells (including tumor cells) induce the production of lymphokines and cytokines (interferons, interleukins and tumor necrosis factor) and activate T cells and specific immune T cells cytotoxic to virus-infected cells (including tumor cells). 7. Measles virus may activate suppressor cells and both directly infecting lymphoma cells) and indirectly (by inducing molecular mediators of suppressor mononuclear cells inhibitory to the growth of neoplastic lymphoid hematopoietic cells) induce remissions of lympho- and hematopoietic malignancies. 8. Retroviral vectors deliver genes into tumor cells for encoding new surface antigens that render the tumor cells highly antigenic and more vulnerable to rejection type immune reactions of the host. Examples illustrate statement. Immunotherapy of tumors with active tumor-specific immunization after the induction of suppressor cells by fetal antigens and the elimination of proliferating suppressor clones by cyclophosphamide will again be proposed

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## Viral oncolysates as human tumor vaccines.

Sinkovics JG.

Cancer Institute, St. Joseph's Hospital, Tampa, Florida.

Postoncolytic immunity entails immune reactions acquired through an oncolytic virus infection or through repeated immunizations with viral oncolysates (or virally modified tumor cell membranes) that are valid and operational also against virally not modified tumor cells of the same type. NK cells react to budding virions, induce target cell lysis primarily but not exclusively by the product of granzymes and pore-forming proteins and operate without direction from memory cells. In contrast, immune T cells (including some TIL) are MHC-restricted, under the direction of memory cells and lyse target cells primarily but not exclusively by the release of lymphotoxin (TNF beta) causing programmed cell death (apoptosis) through endonuclease activation and target cell DNA fragmentation. This author proposes that it is not NK, but the immune T cells mediate postoncolytic immunity. Oncogene amplification may protect immortalized tumor cells even when expressing peptide antigens through MHC molecules against lymphotoxin-mediated apoptosis; but virally-infected tumor cells releasing budding virions remain susceptible to NK cells. Highly immunogenic viral oncolysates should present both budding virions for NK cells and processed viral and tumoral peptide antigens co-jointly for immune T cells.

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